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A CONTRIBUTION TO THE CHARACTERIZATION OF THE PATHOMORPHOLOGICAL CHANGES IN PLAGUE-INFECTED GUINEA PIGS IN RELATION TO THE INFECTING DOSE

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A CONTRIBUTION TO THE CHARACTERIZATION OF THE PATHOMORPHOLOGICAL CHANGES IN PLAGUE-INFECTED GUINEA PIGS IN RELATION TO THE INFECTING DOSE

[Following is the translation of an article by I. N. Koturga and T. I. Basalayeva, published in the Russian-language periodical Materialy Nauchnoy Konferentss po Prirodnoy Ochagovosti i Profilaktike Chumy (Materials from the Scientific Conference on the Natural Focalness and Prophylaxis of Plague), Alma-Ata, Feb., 1963, pages 115--116. Translation performed by Sp/7 Charles T. Ostertag, Jr.]

Materials for the present investigation were the internal organs of 140 guinea pigs which were sent to us by Ye. L. Semenova. They were infected with doses of 10, 50, 100, and 10,000 microbial cells of P. pestis 100. The animals were destroyed in 6--24 hours and 2--10 days (daily); besides this, in each group there were guinea pigs which died in 5--9 days.

Following infection with a dose of 10 microbial bodies, general hyperplastic processes on the part of the reticulo-endothelium were observed in the liver, spleen, lungs, and the regional lymph nodes of the slaughtered guinea pigs. Degenerative changes in the form of granular dystrophy and fatty degeneration took place in the livers of animals which were slaughtered in 8--10 days.

Infection with a dose of 50 microbial bodies caused diffuse fatty degeneration in the liver, focal necroses of the hepatic cells and granulomas. The granulomas consisted mainly of epithelioid cells with an admixture of cells of the lymphoid type and neutrophilic leukocytes; the latter were concentrated in the center of the granulomas and were in a state of degeneration. There was a mass of microorganisms among the cells of the granulomas. In the spleen against a background of general hyperplasia of the reticulo-endothelium, from the sixth through the tenth days endothelioid-cellular granulomas and numerous foci of necrosis were detected. In the lungs, during the first to the seventh days there were infiltrative processes in the interalveolar septa and close to the vessels. Later embolismic foci of pneumonia were noted with necrosis of the elements of the exudate, and a tremendous number of microbes.

In the regional lymph nodes, we detected hyperplasia of the reticulo-endothelium, small foci from the multiplying reticular cells, epithelioid-cellular granulomas with leukocytic infiltration in the center.

During infection with a dose of 100 microbial bodies endothelioid-cellular granulomas were recorded in the liver and spleen from the fourth to the fifth days, and by the sixth to seventh days necrosis of their

cellular elements developed, beginning with the center and taking over the entire granuloma. In the lungs by the fourth day, a serous-hemmorrhagic pneumonia took place with a great number of microbes in the exudate. By the seventh day necrosis of the elements of the exudate developed.

In the regional lymph nodes hyperplastic processes were noted, and the formation of granulomas and their necrosis.

Following the introduction of 10 thousand microbial cells, intensive degenerative-necrotic changes were noted in the liver, spleen, lungs, and regional lymph nodes. Proliferative processes were expressed weakly.

In dead guinea pigs intensive fatty degeneration of the hepatic cells, extending to necrosis, was detected in the liver. In places the blood vessels were devoid of endothelial lining. The tunica media and the tunica adventitia were frayed, hemoginized [?] or necrotized and endothelioid-cellular granulomas with partial or complete necrosis were noted. Numerous foci of necrosis, running together in places, were determined in the spleen.

In the center of the necrotic foci a structureless mass was disposed, and along the periphery -- cellular detritus and a multitude of microorganisms. The blood vessels had the above described changes.

In the lungs there was a focal or mixed serous-hemorrhagic pneumonia with massive foci of necrosis, including the elements of exudate, lung tissue and blood vessels.

In the regional lymph nodes there was extensive focal necrosis, including the lymphoid and reticular tissue and the blood vessels.

In generalizing the data obtained it can be said that with an increase of the infecting dose there is an increase in the intensity of the pathomorphological changes and a change in their nature.

Infection with a dose of 10 microbial cells causes proliferative-hyperplastic processes, and with an increase of the dose up to 50 microbial cells there is a clearly expressed tendency for the formation of granulomas and the intensity of degenerative processes is increased.

The introduction of 100 microbial bodies causes the earlier appearance of granulomas and necrobiotic changes in them. With an infecting dose of 10 thousand microbial cells the necrotic changes project out on the first plane.

Apparently the necrotic changes in the liver have a twofold origin: On the one hand this is the result of fatty degeneration (extensive foci of necrobiosis of the hepatic cells), and on the other these are limited foci, developed as the result of necrosis of the granulomas. In the spleen, regional lymph nodes and the lungs, necroses develop at the sites of granulomas and inflammatory foci.

In the present investigation, we used a great deal of experimental material to support the fact of the regular emergence of granulomas during plague in guinea pigs.